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Acute Coronary Syndromes

DECREASED MYOCARDIAL DENDRITIC CELLS IS ASSOCIATED WITH IMPAIRED REPARATIVE FIBROSIS AND DEVELOPMENT OF CARDIAC RUPTURE AFTER MYOCARDIAL INFARCTION IN HUMANS

Poster Contributions

Hall C

Monday, March 31, 2014, 9:45 a.m.-10:30 a.m.

Session Title: Acute Coronary Syndromes: Biologic Considerations

Abstract Category: 1. Acute Coronary Syndromes: Clinical

Presentation Number: 1265-253

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Background: Dendritic cells (DCs) play pivotal roles in regulating the immune system and inflammatory response. We previously reported DCs infiltration in the infarcted heart and its immunoprotective roles in postinfarction healing process with animal myocardial infarction (MI) models. However, its clinical significance has not been determined.

Methods: The extent of DCs infiltration and its correlation with post-MI healing process in the human infarcted heart were investigated in 24 autopsy subjects after ST-elevation MI. Patients were divided into two groups according to the presence ($n = 13$) or absence ($n = 11$) of cardiac rupture (CR), including LV free wall rupture and ventricular septal perforation. The number of infiltrated DCs and macrophages and the extent of reparative fibrosis in the infarcted area were examined via immunohistochemical (IHC) and masson-trichrome (M-T) stainings.

Results: Baseline characteristics of the study patients were comparable between the CR and the non-CR groups including the history of prior MI, the time from onset to death, and the rate of reperfusion therapy. M-T staining showed decreased % area fraction of reparative fibrosis (%AF) in patients with CR compared to those without ($p=0.0008$). IHC of the infarcted myocardium showed an increase in the number of infiltrating CD68+ macrophages ($p=0.0009$), and a decrease in CD209+ DCs ($p=0.0007$), in patients with CR compared to those without. Furthermore, the CD68+/CD209+ cell ratio was higher in patients with CR compared to those without ($p=0.026$). No significant correlation was noted between the number of CD68+ macrophages and %AF in the infarcted area ($r = -0.18$, $p = 0.39$). However, there was a significant positive correlation between the number of CD209+ DCs and %AF ($r = 0.88$, $p < 0.0001$), and a significant inverse correlation between the CD68+/CD209+ cell ratio and %AF in the infarcted area ($r = -0.83$, $p < 0.0001$).

Conclusions: Decreased number of DCs in human infarcted myocardial tissue was associated with increased macrophages infiltration, impaired reparative fibrosis, and the development of CR after MI. These findings suggest the protective role of DCs in the post-MI inflammation and subsequent healing process.